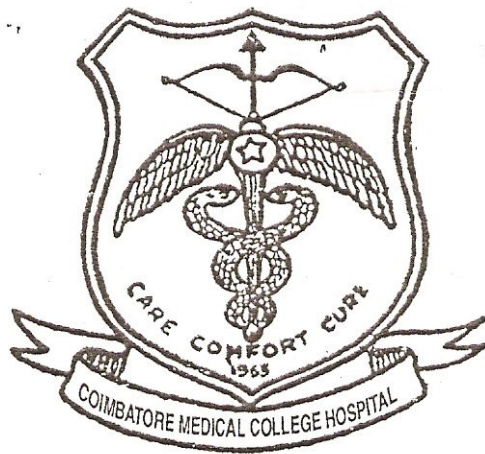


**A STUDY ON BLUNT INJURY TO THE EYE
AND ITS MANAGMENT**

**CONDUCTED IN
COIMBATORE MEDICAL COLLEGE AND HOSPITAL**



**DISSERTATION SUBMITTED TO
THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY IN
PARTIAL FULFILLMENT OF THE REGULATION FOR
M.S. DEGREE IN OPHTHALMOLOGY**

MARCH - 2008

DECLARATION

*I solemnly declare that the dissertation titled “A STUDY ON BLUNT INJURY TO THE EYE AND ITS MANAGEMENT” was done by me at **Coimbatore Medical College and Hospital** during the period from January 2006 to October 2007 under the guidance and supervision of* **Prof. Dr. V.R.Vijaya Raghavan.**

*This dissertation is submitted to the **Tamil Nadu Dr. M.G.R. Medical University** towards the partial fulfillment of the regulation for the award of **M.S. Degree in Ophthalmology.***

Place: Coimbatore

Dr. P. SUMATHI

Date:

DEPARTMENT OF OPHTHALMOLOGY
COIMBATORE MEDICAL COLLEGE AND HOSPITAL
COIMBATORE

CERTIFICATE

This is to certify that the Dissertation entitled “A STUDY ON BLUNT INJURY TO THE EYE AND ITS MANAGEMENT” is a bonafide work of Dr. P. SUMATHI, Post Graduate in Ophthalmology, Coimbatore Medical College. The thesis work has been prepared by her under my guidance and supervision from January 2006 to October 2007 and this dissertation is submitted to the Tamil Nadu Dr. M.G.R Medical University in partial fulfillment of the regulation for the award of Degree of M.S. in Ophthalmology.

Dr. HEMALATHA GANAPATHY M.D.
DEAN
COIMBATORE MEDICAL COLLEGE

PROF. DR. V.R. VIJAYARAGHAVAN
MS, DO, DNB, FRCS.
PROF. AND HEAD
DEPARTMENT OF OPHTHALMOLOGY
COIMBATORE MEDICAL COLLEGE

ACKNOWLEDGEMENT

I am very grateful to **Prof. HEMALATHA GANAPATHY M.D** Dean, Coimbatore Medical College for granting me permission to do my dissertation at Coimbatore Medical College.

I express me sincere thanks to **Prof. V.R. VIJAYARAGHAVAN M.S, D.O, FRCS**, Head of the Department, Ophthalmology for his valuable guidance, and help provided throughout the study.

My sincere thanks to **Prof. A. RAJENDRA PRASAD M.S, D.O** for his encouragement and guidance to contact the study. Special thanks for his guidance to take photographs, without his help the excellent photographs in the study could not have come up.

I express my sincere thanks to **Prof. K. MARAGATHAM M.S, D.O** who initiated the study.

My sincere thanks to **Dr. J. SARAVANANAN M.S** Ophthalmology, **Dr. P. SHANTHI M.S**, Ophthalmology and **Dr. PARAMASIVAM D.O** who gave final shape to the study.

Finally I am very grateful to all my patients for their co-operation, if not this study could not have been possible.

CONTENTS

	Page No
PART I	
INTRODUCTION	1
REVIEW OF LITERATURE	2
PART II	
AIM OF THE STUDY	34
MATERIALS AND METHODS	35
OBSERVATIONS	36
DISCUSSION	54
SUMMARY	64
CONCLUSION	68
BIBLIOGRAPHY	
PART III	
PROFORMA	
MASTER CHART	
INDEX TO MASTER CHART	

A STUDY ON BLUNT INJURY TO THE EYE AND ITS MANAGEMENT

Introduction

Ocular trauma is one of the major causes of visual impairment worldwide. Annually there are in excess of 2 million cases of ocular trauma with more than 40,000 cases sustaining significant visual impairment on a permanent basis. The incidence of ocular injuries are on the rise with increased industrialization and modernisation.

Blunt ocular trauma is more common among mechanical injuries to the eye. Blunt injuries occur at work place, home, in sports, motor vehicle accident, assault etc.

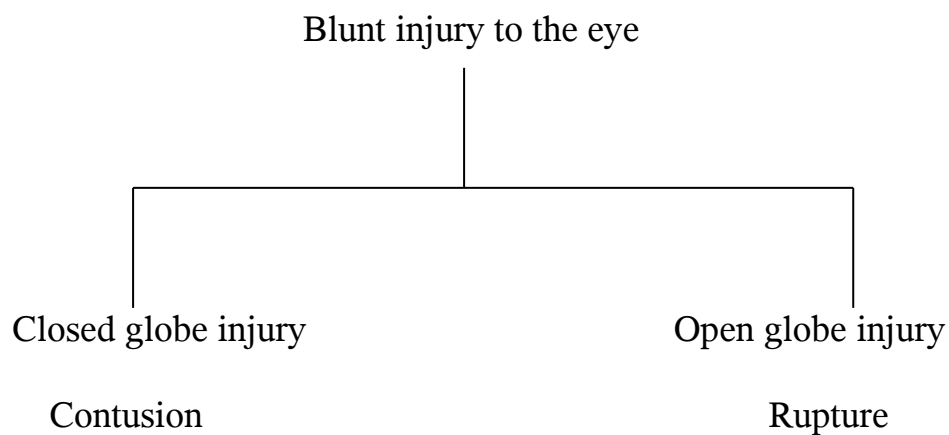
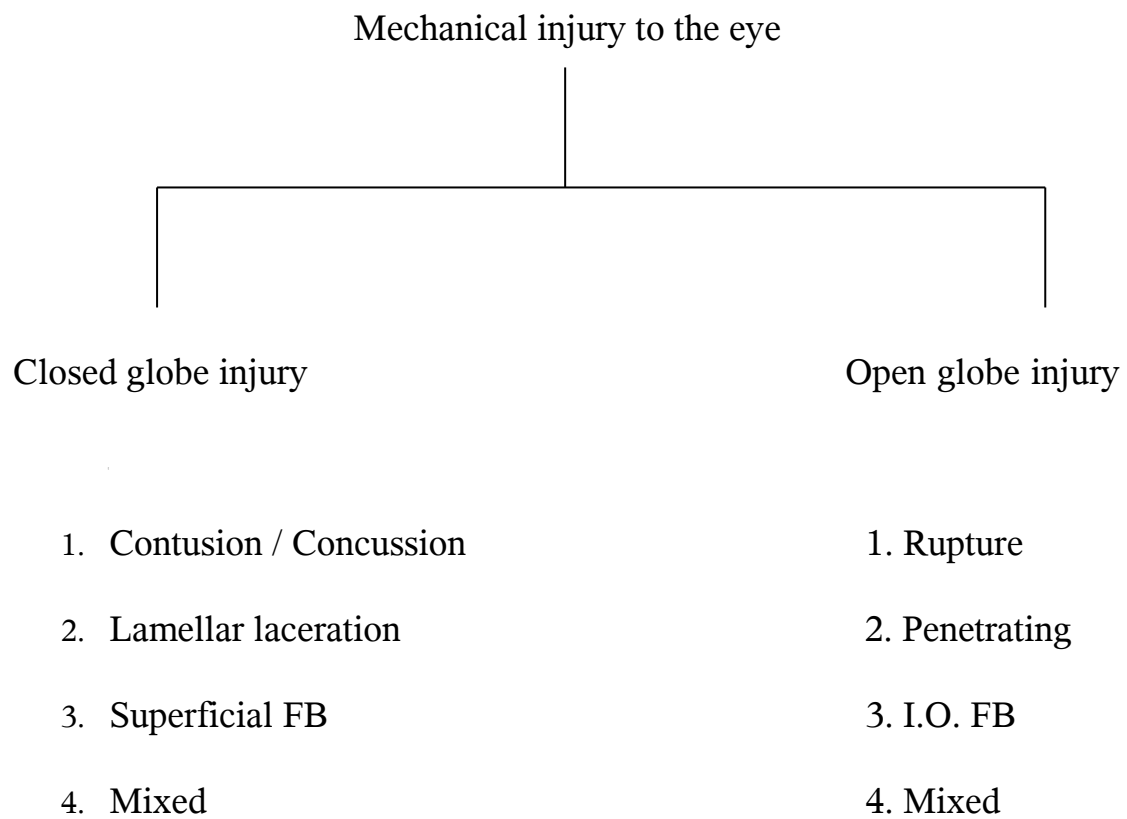
Despite the natural protection afforded to the globe by means of the position of the eye ball in a strong bony orbit composed of the skull and frontal bones, orbital soft tissues and the physiological blink reflex. Eye trauma though trivial has a great significance.

Ocular trauma not only causes visual impairment but also levies a tremendous financial penalty. In the past, severe blunt trauma invariably resulted in devastating visual loss. Understanding the pathophysiologic mechanism and advent of microsurgical technique and instrumentation has greatly improved the visual prognosis.

REVIEW OF LITERATURE

BLUNT INJURY EYE

Ocular injuries can be classified into mechanical and non mechanical injuries. Mechanical injuries can be blunt injury or penetrating injury to the eye.



Ocular injury classification^{T1, J1}

Open globe Injury	Closed globe Injury
Type: <ol style="list-style-type: none"> 1. Rupture 2. Penetrating 3. Perforating 4. Intra ocular Foreign body 5. Mixed Grade <ol style="list-style-type: none"> 1. Visual acuity (6m) 2. $\geq 20/40$ 3. 20/50 to 20/100 4. 19/100 to 5/200 5. 4/200 to light perception 6. No light perception Pupil: Positive: Relative afferent pupillary defect present. Negative: Relative afferent pupillary defect absent.	Type: <ol style="list-style-type: none"> 1. Contusion / Concussion 2. Lamellar laceration 3. Superficial Foreign Body 4. Mixed Grade <ol style="list-style-type: none"> 1. $\geq 20/40$ 2. 20/50 to 20/100 3. 19/100 to 5/200 4. 4/200 to light perception 5. No light perception Pupil: Positive: Relative afferent pupillary defect present. Negative: Relative afferent pupillary defect absent.
Zone: <ol style="list-style-type: none"> 1. Isolated to cornea 2. Corneoscleral limbus to a point 5mm posterior to sclera. 3. Posterior to the anterior 5mm of sclera 	Zone: <ol style="list-style-type: none"> 1. External (bulbar conjunctiva, sclera, cornea) 2. Anterior segment (internal to cornea and including the posterior lens capsule and pars plicata). 3. Posterior segment (posterior to

	posterior lens capsule).
--	--------------------------

Contusion is a closed globe injury resulting from a blunt object.

Injuries occur either at the site of impact or at a distant site secondary to changes in globe configuration or momentary intra ocular pressure elevation.

Mechanism

1. Coup and contre coup:

Direct injury is called coup injury and indirect injury is called contre coup injury. Courville^{J2} introduced the concept of coup and contre coup injury to explain brain damage caused by blunt trauma to head. Wolter^{J3} (1963) adapting Courville's mechanism of brain contre coup injury concluded that a similar injury to the eye could be explained by a line of force traversing the eye causing damage at the interfaces. The extent of such damage depends on the force applied. The most posterior interface between the retina and choroid, choroid and sclera being most commonly involved.

2. Anteroposterior compression and equatorial expansion-by Arlt (1963):^{T5}

The volume of a closed space cannot be changed. The impinging force acts anteroposteriorly forcibly expanding the globe around the equator to the line of impact. The cornea could be indented upto 8.5mm reducing the original anteroposterior axis by 41% and bringing the

posterior surface of the cornea into contact with the iris and the lens. The equatorial plane could be expanded to about 128% of its original length. But before it recovers its normal shape there can be an overshoot and the resulting rapid deformation of the posterior pole and vitreous, can lead to injury, remote from the impact site producing contre-coup injury.

INJURY TO CONJUNCTIVA

Conjunctival hemorrhage is the most common accompaniment of ocular trauma. It can vary from small petechia to large extravasation. Subconjunctival hemorrhage from orbital bleeding does not reach anteriorly upto limbus and its posterior limit cannot be made out. Severe orbital bleeding leads to ballooning of conjunctiva between the lids and must be kept lubricated with ointment covered with a plastic film until the swelling subsides and the conjunctiva returns within the fissure. Topical application of a rubber globe filled with crushed ice is a good method for minimizing the swelling in the acute phase.

Conjunctival chemosis:

Chemosis are conjunctival oedema can occur with or without hemorrhage. It is often a serious portent. The area of maximum chemosis often is a clue to the location of a scleral rupture or perforation. Conjunctival crepitus is air under the conjunctiva. It is usually associated with fracture of lamina papyracea of the ethmoid or other paranasal sinuses and lacerations of their mucosa ^{T2}.

Conjunctival laceration:

Every conjunctival laceration potentially overlies and may conceal a scleral laceration or rupture ^{T2}. Associated conjunctival hemorrhage and oedema may obscure the presence of transparent vitreous, black uveal tissue or gray slimy retina, any of which may have herniated through a scleral laceration. So almost every conjunctival laceration should be examined carefully. Dissection of conjunctiva and Tenon's fascia off the scleral wall determine whether it has been lacerated, punctured or ruptured.

Conjunctival lacerations heal rapidly. Surgical repair is not required for lesions of size less than 1cm. Larger conjunctival tears are sutured with 6-0 or 7-0 gut sutures. During surgical reapproximation care must be taken to avoid inclusion of tenon's fascia in the wound. If it is included it leads to a chalky white herniation. If the conjunctival edges are rolled or inverted while surgery it leads to the development of an inclusion cyst.

INJURY TO CORNEA**Corneal abrasion:**

Loss of epithelium of cornea is called corneal abrasion. It is highly painful and is associated with photophobia. It stains with fluorescein dye. It has a sharp edge. Abrasion is treated with topical antibiotic drops and

cycloplegics for comfort. Eye ointments are to be avoided as they delay wound healing. Patching or bandage contact lenses are used in large abrasions. Small abrasions may be left unpatched. Topical anaesthetic agents should never be^{J4} prescribed because they compromise epithelial wound healing.

Corneal endothelial damage is more serious. A local concussion can rupture endothelial cells and loosen the intercellular tight junctions. The transient corneal oedema usually clears, but endothelial damage can be permanent.

Recurrent corneal epithelial erosion:

Any shearing injury may damage epithelial basement membrane adhesion complexes and consequently lead to recurrent or persistent epithelial defect^{T1, J5}. Patient will typically have an acute onset of pain, redness and tearing on awakening.

Treatment consists of gentle debridement of devitalized, disadhesive epithelium and debris with a moistened cotton tipped applicator or cellulose sponge. Jeweller's forceps may be used to remove loose epithelial edges carefully. A pressure patch is applied until the epithelium is healed. Preservative free lubricants are continued for 6 to 8 weeks. If this is not effective a therapeutic bandage contact lens may be applied and worn continuously for months which allow adequate

adhesion.

Surgical intervention is needed in cases unresponsive to medical therapy. For cases with abnormal basement membrane, superficial keratectomy can be performed. Recurrent erosion without sub epithelial abnormality is treated with anterior stromal micro puncture^{J6}. It is done with a 21-gauge needle^{T1}. Multiple micro punctures through Bowman's layer into anterior stroma are made. This stimulates micro cicatrisation between epithelium and underlying tissue. It is reserved for small erosions outside the visual axis. Phototherapeutic keratectomy with 193 nm excimer laser can be used for recurrent erosion^{T9}. Zone of ablation is 6 -9 mm, ablation depth 10-30micron.

Descemet's tear:

Descemet membrane tear is common. Long sinuous or crescentic tear are usually noted. Some times as multiple short dehiscences or falciform detachment, floating in the anterior chamber can occur. Pigmentary deposits on the posterior surface of the cornea can also occur after contusion.

Blood staining of cornea:

It occurs when blunt injury is associated with massive hyphaema and raised intraocular pressure. The damaged corneal endothelium permits breakdown products of RBC to enter the stroma. So it is the erythrocyte breakdown products and hemosiderin in keratocyte ^{T1}. At first

it is rusty brown or greenish black. And then gradually changes to greenish yellow or grey. It may be in the form of a central ring or disc. Clearing starts from the periphery and takes months to years for complete clearing. Central cornea is the last to clear and can lead to permanent visual impairment. In very young children it might lead to deprivation amblyopia. So it is important to evacuate the hyphaema if associated with elevated intra ocular pressure at the earliest.

Traumatic hyphaema:

Hyphaema is the presence of blood in anterior chamber. It is graded by the volume of anterior chamber filled with blood after layering of red blood cells^{T1}.

Grade I : Less than one third of anterior chamber.

Grade II : One third to one half of anterior chamber.

Grade III: One half to nearly total.

Grade IV: Total (Eight ball).

Microscopic hyphaema occurs in small anterior chamber bleeds without actual layering of blood but with evidence of suspended red blood cells on slit-lamp examination.

Bleeding usually follow a tear in anterior face of ciliary body, iris, cyclodialysis and iridodialysis. Bleeding usually stops as a result of tamponade by increased intra ocular pressure, vascular spasm, and

formation of fibrin platelet clot. Maximal clot integrity is achieved within 4-7 days after injury. Unlike clots in other parts of the body hyphaema demonstrate no fibroblastic activity or neovascularisation.

Clot breakdown occurs as a result of fibrinolytically active anterior chamber. The free blood cells and fibrin degradation products are then cleared through the normal trabecular outflow pathways. Absorption through iris vasculature may have a minor role. Rebleeding occurs in about 20% of cases^{T2} and seen as bright red blood, layered over darker clotted blood and increased hyphaema size. It occurs 2 to 5 days after injury due to clot lysis and retraction. Teboul and colleagues reported lower incidence of rebleeding in paediatric patients^{J7}.

Small hyphaemas clear spontaneously. Large hyphaemas may fail to clear and its colour changes to black. It is called eight ball hyphaema. It is invariably associated with increased intraocular pressure. Children under 6 years of age are most susceptible to visual loss^{T2}. Surgical intervention is done to prevent major complications like blood staining of cornea, peripheral anterior synechiae and optic atrophy secondary to glaucoma.

Blood staining of cornea occurs if intra ocular pressure >25mm Hg persists for atleast 6 days. Optic nerve damage can occur if intraocular

pressure is >50mm Hg for atleast 5 days or 35mm Hg for atleast 7 days. Patients with sickle cell disease are at even greater risk due to their lesser ocular tolerance due to blood flow impairment. Peripheral anterior synechiae in unoperated eyes can occur if hyphaema persists for 9 days or more.

Surgical management is with paracentesis with anterior chamber wash. If blood is clotted, then a larger incision is made with clot extraction by gentle pressure at the limbus. Rebleeding can be minimized with antifibrinolytic agents like amino caproic acid orally 50mg/kg every 4 hours upto 30gm / day for 5 days ^{T1}. Cessation of the drug before 5 days has a greatest chance for rebleeding^{J8}.

Effect of blunt injury on intra ocular pressure:

Immediately after injury intra ocular pressure may be normal, increased or decreased. Aqueous out flow may be decreased because of angle recession, inflammatory trabeculitis or hyphema. Ciliary body injury tends to decrease aqueous inflow so leads to decreased intra ocular pressure. Pressure recording is very important in large hyphema especially in sickle-cell disease central retinal artery occlusion can occur even if the pressure is only in the high twenties or low thirties.

Late glaucoma can occur because of obstruction of trabecular

meshwork with proliferating corneal endothelial cells or descemet's membrane, proliferation of fibroblasts and peripheral anterior synechiae probably due to inflammation or from organization of blood in the angle for more than 7 days^{T1}. Other causes are due to direct damage to trabecular meshwork, angle recession, ghost cell glaucoma. Treatment of acute glaucoma with drugs is needed when IOP>40mm Hg and for lasting IOP> 30mm Hg for 2 weeks or more.

Aqueous suppressants such as topical Beta blockers and oral carbonic anhydrase inhibitors are used. Miotics, prostaglandin analogues and epinephrine drops are to be avoided because of their inflammatory potential^{T6}. Topical atropine reduces the risk of secondary hemorrhage by stabilising blood ocular barrier. In sickle cell disease, avoid drugs like carbonic anhydrase, alpha adrenergic agonists and mannitol are to be avoided because they can lead to hemo concentration and sickling. Trans corneal oxygen therapy has be tried to decrease the intra ocular pressure^{T6}. By increasing the partial pressure of oxygen, cells in the anterior chamber are less likely to sickle. Surgical intervention is needed if medical management fails.

INJURY TO IRIS

Traumatic iritis:

Mild inflammation of iris and ciliary body may occur following

any trauma to the eye. IOP is lower than normal in early post traumatic period. Aqueous humour contains cells and fibrin. It responds to topical steroids and cyloplegics. After severe injury plastic iridocyclitis may develop which is resistant to treatment.

Traumatic miosis and mydriasis:

Miosis occurs immediately after injury.

Mydriasis present with dilated pupil and slightly irregular. Hard blow leads to rupture of the iris sphincter and permanent deformity of the pupil. Small tears are left alone. Large tears are sutured by MC canal's suture^{T8} technique. It can lead to areas of atrophy causing transillumination defect due to pigment liberation.

Traumatic Iridodialysis:

Disinsertion of iris root from the ciliary body is called iridodialysis. It can lead to uniocular diplopia. Large dialysis are repaired with sutures brought out through the ciliary sulcus^{J9}.

Traumatic angle recession:

Angle recession occurs when the longitudinal fibres of the ciliary muscle detaches from the scleral spur. Clinically it presents with wide angle. It may lead to late onset secondary open angle glaucoma. It is due to fibrosis of trabecular meshwork. 20-100% of eyes sustaining traumatic hyphaema have angle recession^{T2}. Deep angle tears are visualized with

ultra sound bio-microscope. If angle recession is > 180 degree, then there is more chance for developing glaucoma^{T2}. If surgery is needed more chance of failure with trabeculectomy can occur, so anti metabolites are to be added^{T6, J10}.

Traumatic aniridia:

If the root of the iris is completely torn from its attachment to the ciliary body it leads to traumatic aniridia. It occurs in gross injury causing severe damage to other ocular tissues and hyphaema is the rule.

INJURY TO THE LENS AND ZONULES

Subluxation and dislocation of lens:

Blunt trauma to the globe can break the zonular fibres that encircle the lens radially and anchor it to the ciliary body. When 25% or more of the zonules are broken it leads to subluxation^{T2}. Subluxation is far more frequent than dislocation and produces visual problems like astigmatism which cannot be corrected with the spectacles or contact lens unocular diplopia occurs if the equator of the lens crosses pupillary zone (lateral subluxation).

Refractive correction of phakic or aphakic portion to be done. If visual acuity with refractive correction is good, spectacles are prescribed. Surgery is indicated in severe subluxation.

Dislocation of lens:

A dislocated or luxated lens is a lens that is completely detached from its zonular and vitreous attachment. It can dislocate into anterior chamber or into vitreous. Dislocation into vitreous is more common than into anterior chamber. Emergency surgery is needed for anterior dislocation of lens incarcerated in the pupil causing glaucoma^{T2}. Because both the condition leads to elevated intra ocular pressure. In anteriorly dislocated lens, lens removal is the procedure of choice after vitrectomy.

Posteriorly dislocated lens can lead to severe iridocyclitis and secondary glaucoma can occur in 90% of cases although it may not be evident for many years. At first the lens is mobile in the vitreous (lens natans) on prone position it might enter the anterior chamber as a wandering lens but eventually organized membrane tend to anchor it. Posteriorly dislocated lens is left alone, rarely pars plana vitrectomy with nucleus removal is needed.

Rarely the lens can slip through retinal tears into intra retinal space or can lie between sclera and ciliary body as subsclearal luxation. In severe contusion associated with globe rupture lens can lie beneath intact conjunctiva as phacocoele. Suspect marfan's syndrome weill marchesani syndrome, syphilis and retinitis pigmentosa^{T6} in case of subluxation or

dislocation of lens after a trivial trauma.

Vossious ring:

A circular ring of faint or stippled opacity is seen on the anterior surface of the lens due to the pigments from the pupillary ruff being imprinted on the anterior surface of the lens. The size of the ring corresponds to the size of constricted pupil. It is seen only after dilatation of pupil. It is visually insignificant and gradually resolves with time, it serves as an indicator of prior blunt trauma^{T10}.

Traumatic cataract:

It is partly due to the mechanical effects of the injury on the lens fibres and mainly due to entrance of aqueous occurs following damage to the capsule. If the tear is small and peripherally located, it may not be clinically visible. Tear is most common at the posterior pole which is the thinnest portion of the lens capsule. If the tear is covered by iris, it seals the tear with fibrin at first and later^{T3} by proliferation of sub capsular epithelium which secretes a new capsule. In these cases the entrance of aqueous is stopped and the opacity in the lens may remain stationary or even regress. If the tear remains open, opacification progresses to involve the entire lens. Most typical contusion cataract is rosette shaped cataract^{T10} it usually involves the posterior cortex. The star shaped cortical sutures are delineated and feathery

lines of opacities outlining the lens fibres radiate from them. Rupture of lens capsule leads to leakage of lens proteins into the aqueous and vitreous, it can cause uveitis and / or glaucoma. Care must be taken to rule out the possibility of fibrin covering a clear lens that can masquerade as cataract.

Surgery is indicated only in cases of visual disturbance. Posterior capsule integrity should be assessed before surgery with ultrasound Bscan or ultra sound Biomicroscopy^{T3}.

Indications for surgery:

- ♦ Unacceptable decrease in vision.
- ♦ Obstructed view to assess posterior segment.
- ♦ Lens induced inflammation/glaucoma (Phacolytic glaucoma)^{J11}
- ♦ Capsule rupture with lens swelling (phacomorphic glaucoma).

If posterior capsule and zonules are intact, phacoemulsification with IOL implantation is done in the capsular bag. If zonular dialysis <180° ECCE is done and IOL is placed in the bag with the help of capsular tension ring^{T10}. If posterior capsule is ruptured but zonular support is maintained then sulcus fixation of IOL is done. If capsule and zonular support are insufficient and angle is damaged minimally then scleral fixation is done. If no posterior support then IOL is placed in the anterior chamber.

In young children and in patients with high inflammation, aphakia in the primary sitting is better. Primary lens removal is done if the lens is fragmented, swollen or develops pupillary block. Advantage of secondary cataract removal i.e., after subsidence of inflammation due to its better visibility, better IOL calculation, anterior segments reconstruction and stabilization of hemato ocular barrier.

Post operative complication:

Early complications are corneal oedema, fibrinous uveitis, increased intra ocular pressure, hyphaema. Late complications are posterior capsular opacification, pupillary capture, IOL decentration and retinal detachment

INJURY TO SCLERA

Scleral rupture (globe rupture) is an inside out full thickness tear of sclera ^{T3}. When the overlying conjunctiva remains intact it is called occult scleral rupture.

Most common quadrant of tear is superonasal^{T2} because of the exposure of eye to a glancing blow in the lower and outer parts of the orbit where the globe is least protected, leading to an indirect globe rupture. Direct globe rupture occurs at the point of impact of an impinging force.

Rupture most commonly occurs in a circumferential arc parallel to the limbus 2 to 4 mm behind corneoscleral junction. Rupture occurs opposite to the site of impact at the level of schlemm's canal, at the insertion of rectus muscle on the globe or at the equator^{T2}. They are mostly single. Tear in the sclera appears everted with gaping edges, through which black uveal tissue, retinal tissue protrudes. Anterior chamber is filled with blood. Eye is soft.

Occult scleral rupture is suspected when hemorrhagic chemosis, total hyphema, deep anterior chamber, decreased intra ocular pressure, no perception of light and duction of globe is reduced towards the quadrant of rupture. Primary enucleation is indicated only for an injury so devastating that restoration of anatomy is impossible, and with high risk for sympathetic ophthalmitis. All other cases are surgically repaired.

Repair of globe rupture:

General anaesthesia is almost always required^{T9} because retrobulbar or peribulbar anaesthetic injection increases orbital pressure, which may cause or exacerbate the extrusion of intra ocular contents.

Corneal component is approached first^{T9}. If vitreous or lens fragments have prolapsed through the wound it should be cut flush

with the cornea. Uveal or retinal tissue if present can be repositioned. Frankly necrotic uveal tissue is excised. Corneal laceration at limbus is sutured first, with 10-0 nylon and then remaining corneal tear is sutured.

Scleral component of laceration is approached with peritomy. Conjunctival separation is done only as necessary to expose the wound. Prolapsed vitreous is excised and prolapsed nonnecrotic uvea and retina are repositioned with spatula. Scleral wound is closed with 9-0 nylon or 8-0⁹ silk suture. Conjunctival suture, should not be placed directly over the corneoscleral line of suture^{T8}. Sub conjunctival antibiotics and steroids to be given. Periocular anaesthetic injection may be used to control post operative pain. Corneal suture are left in place for at least 3 months and then removed incrementally over the next few months.

INJURY TO VITREOUS

Vitreous detachment:

Vitreous is disorganised by anterior or posterior detachment or both. It presents as fine pigmentary opacities. Disinsertion occurs at the vitreous base, optic nerve head, retinal vessels, lattice degeneration or chorioretinal scars. Avulsed vitreous base has the appearance of a loose clothesline, a hammock or a ribbon suspended loosely through the vitreous cavity. It is pathognomonic of blunt trauma. Most common site

of vitreous dialysis is infero temporal.

Vitreous hemorrhage:

It occurs due to acute posterior vitreous detachment, avulsion of superficial retinal vessels, retinal tear from torn choroidal or ciliary body vessels. Presence of pigment epithelial cells is an indication for careful search for retinal tear or dialysis.

Indirect ophthalmoscopic examination is done to know the source of hemorrhage. If hemorrhage is dense then ultra-sound Bscan is done. It might reveal a retinal tear or detachment, choroidal detachment, posterior vitreous detachment or occult ruptured globe. Uncomplicated vitreous hemorrhage treated with bed rest and head end elevation and observation for 6 months. Early vitrectomy is indicated if vision loss is bilateral or if retinal detachment is present.

Complication:

Vitreous hemorrhage may get organized by fibrous tissue to form proliferative retinopathy associated with damage to choroid and retina may lead to retinal detachment. Massive hemorrhage can lead to ghost cell glaucoma.

INJURY TO RETINA

Traumatic retinal tear:

Traumatic traction retinal tears occur when the vitreous is violently

shifted away from the retina. -Any area of strong vitreoretinal adhesion is likely to be the site of retinal tear. Concentric tearing around the optic nerve head has been described^{T1, J12}. Traumatic breaks are often multiple, commonly found in the inferotemporal and superonasal quadrants. Tears are usually large, ragged equatorial breaks, dialysis or a macular hole. Less common type of breaks are horse-shoe shaped tears and operculated holes.

Retinal dialysis:

Dialysis is disinsertion of retina from the ora serrata. Common site is inferotemporal and superonasal. Retinal detachment can develop as a late sequelae. It is more common with superonasal dialysis due to gravity. Retinal dialysis without retinal detachment is treated with laser or cryotherapy. In the presence of retinal detachment, scleral buckling is done and reattachment rate is 98%^{T1, J13}.

Retinal detachment:

Blunt trauma is the leading cause of retinal detachment in children and adolescent. Young patients have formed vitreous so detachment progresses slowly, unless a giant tear is present. Retinal detachment is late sequelae of injury. Examination reveals demarcation lines, atrophy of underlying pigment epithelium, subretinal precipitates, retinal macrocysts and extensive vitreous tobacco dust. Proliferative vitreoretinopathy is

uncommon^{T1}, so prognosis for reattachment is excellent. Retinal detachment immediately after contusion occurs in antecedent vitreoretinal pathology.

Traumatic retinal detachment is managed with pneumatic retinopexy, scleral buckling or vitrectomy depending on the location of retinal break and the degree of proliferative vitreoretinopathy ^{T1}. Early reattachment is desirable because apoptotic photoreceptor cell degeneration begins as early as eight hours after traumatic detachment ^{T1}.

Commotio retinae:

Blunt trauma to retina leads to cloudy swelling. It is most commonly seen in the posterior pole, rarely in the periphery. Mechanism for retinal opacification are extra cellular oedema, glial swelling and photoreceptor outer segment disruption. Macular involvement presents with a cherry red spot. Visual acuity may be unaffected or profoundly decreased to 20/400 level^{T6}. Retinal pigment epithelial abnormalities seen by early fluorescein angiography are associated with slow visual recovery. Prognosis for visual recovery is good, as the condition clears in 3-4 weeks. In some cases visual recovery is limited by associated macular pigment epitheliopathy, choroidal rupture or hole formation. FFA does not demonstrate hyperfluorescence from extra vascular fluorescence accumulation. No fluorescein photo confirmation of disruption of blood

retinal barrier. Foveal densitometry in humans demonstrates disruption of photo receptor – retinal pigment epithelium complex^{T1, J14}. There is no acute treatment.

Traumatic macular cyst and holes:

Fovea is the thinnest part of the retina. Blunt trauma can cause full-thickness macular hole by either one or a combination of mechanisms, including contusion necrosis and vitreous traction.

Holes may be noted immediately or soon after injury that cause severe Berlin oedema, after a subretinal hemorrhage caused by a choroidal rupture, following severe cystoid macular oedema or after a whiplash separation of the vitreous from the retina^{T7}. Traumatic macular holes may be successfully closed with vitrectomy and gas injection^{T7}.

Traumatic retinal hemorrhage:

Retinal, subretinal and preretinal hemorrhage are also common after concussion injury. They are of minimal significance if in the periphery. Hemorrhage involving macular area can lead to permanent dysfunction.

INJURY TO CHOROID

Choroidal rupture:

Choroidal rupture occurs with severe form of blunt trauma.

Rupture may occur directly at the site of application of the force or indirectly on the opposite side of the globe. It is due to anteroposterior compression and equatorial expansion^{T6}. The retina is relatively inelastic, and the sclera is relatively tough, so resist rupture. Bruch's membrane on the other hand is inelastic and more prone to rupture^{T6}. The overlying retinal pigment epithelium and underlying choriocapillaries are also torn but in most cases the deep choroidal blood vessels remain intact. Patients with angioid streaks are vulnerable to choroidal rupture. Choroidal rupture may be single or multiple, occurs commonly in the periphery and concentric to the disc. Choroidal rupture involving the fovea is associated with poor visual prognosis. Complication of choroidal rupture is the development of choroidal neovascularisation^{T7}. Most subretinal new vessels regress spontaneously so laser therapy is usually unnecessary.

Patients with choroidal rupture close to the macula is advised to use Amsler grid^{T7} for self testing for early diagnosis of subretinal neovascularisation. Laser photocoagulation using green yellow or red wavelength destroys subretinal neovascular membranes, but recurrences are possible. In case of recurrence subfoveal surgery is considered.

Chorio retinitis sclopetaria:

Combined choroidal and retinal rupture is called chorioretinitis sclopetaria. It occurs with high speed missile injuries^{T7} to the orbit. Large

areas of choroidal and retinal rupture and necrosis are combined with extensive subretinal and retinal hemorrhage often involving as much as 2 quadrants of the retina. As the blood resorbs, the injured area is filled in by extensive scar formation and wide spread pigmentary alteration occurs. Macula is almost always involved^{T7}, leading to significant visual loss, but secondary retinal detachment rarely develops^{T6}.

Traumatic retinal vascular occlusion:

Blunt trauma can rarely lead to generalized retinal vascular constriction and actual occlusion of central retinal vein and central retinal artery.

Purtscher's retinopathy:

Severe head and chest trauma leads to purtscher's retinopathy^{T1}. Patchy whitening of inner retinal layers with retinal hemorrhage and macular sparing is seen during the first 24 hrs. Hemorrhage and retinal whitening clear spontaneously often leaving no visual sequelae. Occasionally patients present with disc oedema and afferent pupillary defect. Vision may be permanently lost from this infarction and optic atrophy may develop.

INJURY TO OPTIC NERVE

Optic disc avulsion:

Backward displacement of optic nerve from the scleral canal is

optic disc avulsion. Avulsion occurs either due to extreme rotation and forward displacement of the globe, backward pull on the optic nerve or sudden increase in intra ocular pressure, causing rupture of lamina cribrosa^{T7}. Total visual loss usually occurs.

Traumatic optic neuropathy:

Traumatic optic neuropathy commonly follows injury to forehead. The impact transmits shock waves to optic canal damaging the optic nerves by shearing action. It can be due to direct trauma to the optic nerve by the fractured bone spicule of optic canal or compression by intrasheath hemorrhage or oedema. Fracture of the optic canal after head injury is characterized typically by a wound at the lateral part of eye brow, associated with epistaxis, loss or diminution of direct light reaction in the homolateral pupil and monocular hemianopic or other field defects. Optic disc pallor develop after 2-3 months. Treatment with high dose steroids started within 8 hours is advised, in the presence of intra sheath hematoma, surgical decompression of optic nerve sheath is tried, but visual outcome is poor.

INJURY TO ORBIT

Eyelid contusion:

Blunt trauma to the eyelid and forehead leads to ecchymosis and oedema of eyelid. It is generally innocuous. But it is important to rule out the possibility of trauma to the globe, fracture of orbital wall or basal

skull fracture.

Orbital floor fracture:

Fracture of orbital floor without fracture of inferior orbital rim is indirect fracture or blow out fracture. Blow out fracture can involve orbital floor or medial wall. Floor fracture is more common. Blow out fracture occurs when the orbit is hit by an object larger than the diameter of orbit. It leads to a sudden increase in intra orbital pressure, and the weakest part of floor gives way^{T2}. The weakest part of the floor is the postero medial part of the floor in the maxillary bone close to infra orbital canal.

It presents as ecchymosis, periorbital oedema and subcutaneous emphysema, diplopia with limitation on upgaze, downgaze or both. Limitation of vertical movement of globe is due to entrapment of inferior rectus muscle. A significant limitation of both horizontal and vertical eye movement indicates nerve damage or generalized soft tissue injury. Enophthalmos and ptosis of globe, and Hypoesthesia in the distribution of infra orbital nerve can occur.

Large fractures of orbital floor leads to prolapse of orbital soft tissue into maxillary sinus leading to enophthalmos. In the early stage enophthalmos is masked by orbital oedema, later it becomes apparent as

the orbital oedema subsides and contracture pulls the soft tissue farther into the sinus.

Floor fracture with visual loss indicates injury to the optic nerve or increased orbital pressure causing a compartment syndrome. It is diagnosed with CT scan orbit with coronal and sagittal view. Majority of blow out fractures does not require surgical intervention. It is usually observed for 7 – 10 days to allow the swelling and orbital hemorrhage to subside. Oral prednisolone 1mg / kg / day for 7 days decrease the oedema and limit the risk of long term diplopia from inferior rectus contracture and fibrosis.

Indications for emergency surgery: In paediatric patients with trapped inferior rectus beneath trap door fracture. Attempted ocular excursions may result in bradycardia due to stimulation of oculocardiac reflex^{T4}.

Indications for surgery^{T4}: In the presence of diplopia with limitation of upgaze and / or downgaze within 30 degree of primary position, with a positive traction test result 7-10 days after injury, with radiological confirmation of fracture of floor, enophthalmos exceeding 2mm that is cosmetically unacceptable. Large fracture involving at least half of orbital floor especially associated with medial wall fracture. Surgery to be done ideally within 2 weeks. Late surgery leads to failure of repair because of fibrosis and contracture. Surgical approach is trans conjunctival with lateral cantholysis.

Fracture roof of orbit:

Orbital roof fracture is more common in children^{T4} who have not yet pneumatized the frontal sinus. It presents as periorbital ecchymosis, inferior or axial displacement of the globe. Large defect has pulsation of globe without bruit. Complications include intra cranial injuries, CSF rhinorrhoea, pneumo cephalus, subperiosteal hematoma, ptosis and extraocular muscle imbalance. Young children may develop non displaced linear roof fracture after fairly minor trauma, which present with delayed ecchymosis of upper lid. Most roof fracture do not require repair.^{T4}

Medial wall fracture:

It presents as periorbital subcutaneous emphysema, acute proptosis due to intraorbital emphysema. Restriction of abduction occurs if medial rectus muscle is entrapped. Surgical intervention is seldom necessary unless severe entrapment of medial rectus muscle^{T4}. High dose antibiotics given to prevent orbital cellulitis.

Lateral wall fracture:

Lateral wall fracture is rare. Because it is more solid than other walls and is usually associated with extensive facial damage. Zygomatic-maxillary complex fracture is tripod fracture and can lead to globe ptosis^{T1}.

INVESTIGATION

1. CT scan-orbit and brain:

- ◆ Axial (One mm), coronal (3 mm), slices are useful.
- ◆ CT detects the exact extent of orbital wall fracture as well as soft tissue injuries such as incarceration of orbital contents in the fracture.
- ◆ Provides visualisation of retro orbital space.
- ◆ Easy recognition of intra orbital and intra ocular air as in penetrating trauma and disruption of paranasal sinuses.
- ◆ CT clearly defines most radiolucent foreign bodies like small pieces of stone ,aluminium, lead free glass < 1.5mm and copper and steel of minimal diameter 0.06mm.

2. Ultra Sound – B scan orbit:

Ultra sound B scan is used to diagnose the following lesions.

- ◆ Integrity of the posterior capsule of the lens in traumatic cataract.
- ◆ Subluxation / dislocation of lens
 - Vitreous hemorrhage seen as high density reflection.
 - Posterior vitreous detachment seen as hyper dense line with after movement in kinetic ultra sound.
 - Retinal detachment seen as smooth bright folded membrane

with high reflectivity.

- Total retinal detachment has a triangular shape with insertion at optic disc and ora serrata.
- Tractional retinal detachment has tented configuration.
- Peripheral retinal dialysis noted as disinsertion of peripheral retina from ora serrata.
- Choroidal detachment has a smooth thick dome shaped elevation, with little after movement. 360 degree choroidal detachment seen as kissing choroidalis.
- Scleral tear as irregular contour.

AIM OF THE STUDY

1. To study the impact of blunt injury on various structures of the eye.
2. To study the visual outcome in different injuries.
3. To study the demographic profile of the patient's affected.
4. Early intervention of traumatic cataract.
5. To assess the involvement of posterior segment in trivial anterior segment lesions.
6. Role of the CT scan in orbital injuries.
7. Role of B scan in the initial management of trauma.

MATERIALS AND METHODS

The study was conducted at Coimbatore Medical College hospital, Coimbatore. The cases were taken from the patient's attending Ophthalmology Department and Emergency Department of Coimbatore Medical College. The study period was from Jan 2006 to October 2007.

110 cases of blunt injury were taken up for the study. Cases included those with trivial external injury to gross visual loss.

A detailed history was taken to know the mode of injury and the duration between injury and presentation. Examination started with visual acuity testing with Snellen's chart, anterior segment examination done with slit lamp biomicroscopy. Intraocular pressure recording done after ruling out globe rupture with the help of schiotz tonometer / applanation tonometer Gonioscopy was done in appropriate cases. Posterior segment examination was done with direct and indirect ophthalmoscope. Field charting was carried out in relevant cases.

Investigation like x-ray orbit CT scan, ultrasound B scan were done in needed cases. All the cases were followed at regular intervals to assess the visual outcome and complication.

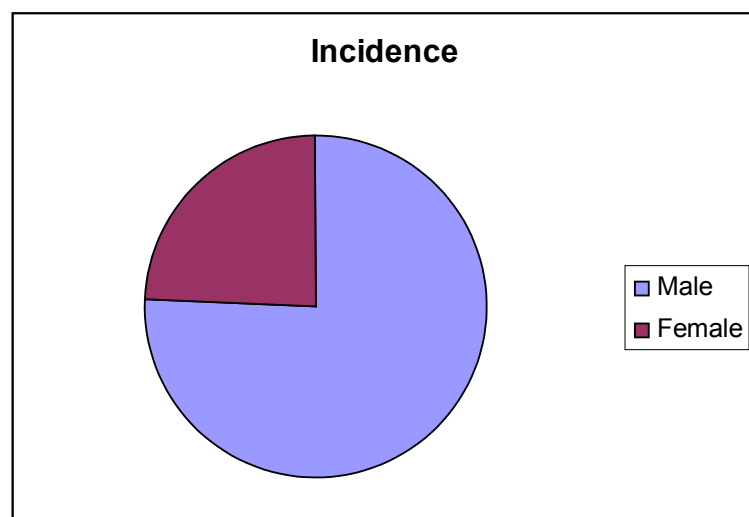
OBSERVATIONS

110 patients with blunt injury to the eye were studied.

Sex incidence:

Out of the 110 patients 83 patients were male, 27 patients were female.

Male patients constituted 75.45% of the total and female patients constituted 24.54%. And male to female ratio was 3:1.



Male = 75.45%

Female = 24.54%

Age incidence:

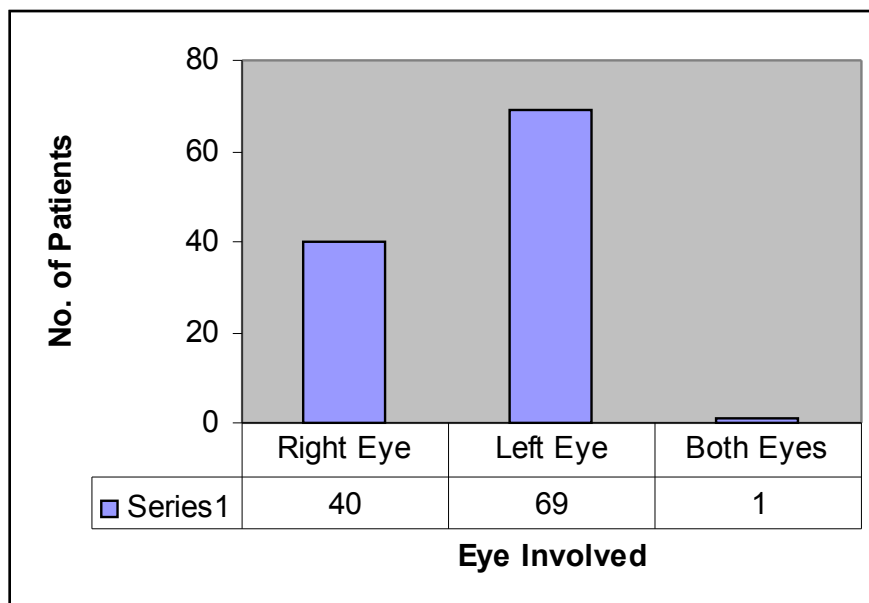
This Study had patients of age ranging from 5 to 70 years. It included both male and female patients.

Incidence of blunt trauma among various age group is as follows.

Age Group	Adult		Children		Total Percentage
	Male	Female	Male	Female	
0 – 10 Yrs	-	-	5	-	4.5%
11 – 20 Yrs	7	-	2	3	10.9%
21 – 30 Yrs	27	7	-	-	30.9%
31 – 40 Yrs	18	7	-	-	22.7%
41 – 50 Yrs	14	4	-	-	16.36%
51 – 60 Yrs	7	5	-	-	10.9%
61 – 70 Yrs	3	1	-	-	3.63%

Maximum number of injuries were seen in the age group of 21 – 30 Years.

Eye Involved:



Mode of injury:

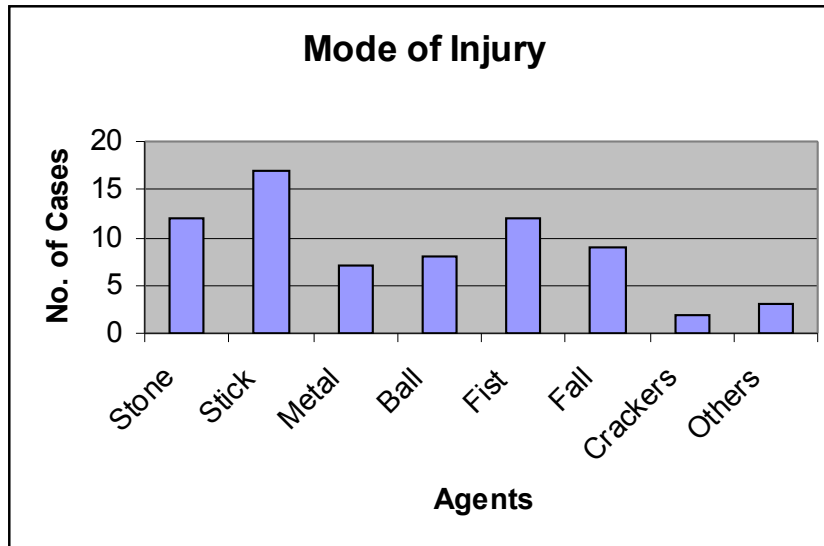
Injury caused by road traffic accidents (RTA) and non- road traffic accidents (non-RTA) were included in this study. Injuries were caused by various agents like stone, stick, metal, ball, crackers, fall by self, fist injury and others.

The total number of RTA cases were 40 and non-RTA cases were 70. The agents causing them are shown in the following Table:

NON – RTA		
AGENT	NO. Of CASES	PERCENTAGE
Stone	12	10.9%
Stick	17	15.45%
Metal	7	6.36%
Ball	8	7.27%
Fist	12	10.9%
Fall	9	8.18%
Crackers	2	1.81%
Others	3	2.72%

RTA cases 36.36 %.

Non RTA cases 63.63 %.



Incidence among various injuries:

RTA	-	36.36%
Work Place Related injuries	-	33.63%
Sports	-	10%
Others	-	20.01%

Among the non RTA patients, injury with stick was the most common injury. Among RTA patients injury due to a fall from a two wheeler, or due to injury by the metallic parts of the two wheeler was more common.

VISION ON PRESENTATION:

Out of 110 patient's vision varied from NoPL to 6/6. 8 patients had NoPL, 33 patients had vision from PL to HM, and 29 patients had vision from 1/60 to 6/60, 19 patients had vision from 6/36 to 6 /18 and 21 patients had vision from 6/12-6/6.

VISION ON FOLLOW UP:

Vision on follow up after 1 month showed, 17 patients with NoPL and all were due to optic nerve injury, 16 patients with vision HM to 2/60, 7 patients with vision 3/60-6/60, 16 patients with vision 6/36-6/18, 54 patients with vision 6/12-6/6.

OCULAR TENSION ON PRESENTATION:

Ocular tension was recorded after ruling out corneal abrasion and globe rupture. Ocular tension was not recorded in 12 patients with corneal abrasion and in 9 patients with globe rupture. 12 patients had intraocular pressure greater than 25mm of Hg due to hyphaema in 7 patients, due to iritis in 4 patients and due to dislocation of lens into anterior chamber in 1 patient. Rest of the patients had normal ocular tension.

Five patients with hyphema responded to aqueous suppressants and 2 patients needed paracentesis and patients with iritis responded to treatment. One case with anterior dislocation of lens was treated with lens removal and pressure returned to normal.

TENSION ON FOLLOW UP:

On follow-up after three months all the patients had normal intraocular pressure except for two patients who went for phthisis bulbi.

ANTERIOR SEGMENT LESION:

Conjunctiva	SCH	51
	Tear	6
Cornea	Oedema	12
	abrasion	8
Anterior chamber	Hyphaema	19
Iris	Iritis	14
Pupil	Miosis	3
	Mydriasis	14
Lens	Cataract	12
	Subluxation	4
	Dislocation	4
Angle of anterior chamber	Recession	1

POSTERIOR SEGMENT LESION:

Vitreous	Hemorrhage	5
Retina	Tear	4
	Detachment	3
Macula	Oedema	4
	Hole	1
ONH	Optic nerve injury	13
	Atrophy	4
	Avulsion	1
Choroid	Tear	4
	Hemorrhage	2

GLOBE RUPTURE: 9 PATIENTS

ORBITAL INJURES:

Nerve Palsy	3 rd nerve	4
	6 th nerve	2
Eye Lid	Ptosis	9
	Lagophthalmos	2
Blow out fracture		6
Medial wall fracture		1
Lateral wall fracture		7
Roof of orbit fracture		2

CONJUNCTIVA:**Sub conjunctival hemorrhage:**

51 patients presented with subconjunctival hemorrhage. Out of this 10 patients had extension of hemorrhage from the orbit following fracture of the orbital walls. Rests of the patients were due to direct trauma to conjunctiva.

Conjunctival chemosis:

5 patients had conjunctival chemosis associated with subconjunctival hemorrhage. Out of this one patient had a large superonasal chemosis, which on exploration, showed occult scleral tear with phacocoele. Tear was sutured after lens removal and vitrectomy.

Conjunctival tear:

6 patients presented with conjunctival tear. 5 patients had a tear of size <1 cm who were treated conservatively. 1 patient had a tear of 2 cm which was sutured with absorbable suture material.

CORNEA:

8 patients with corneal abrasion, were treated with topical antibiotic eye drop and cycloplegics with eye patching. 1 patient had recurrent corneal erosion which was treated with wound debridement and bandage contact lens for 3 months. 12 patients had corneal oedema. Out of this, 8 were due to iritis and 4 were associated with traumatic hyphaema. Corneal oedema subsided after treating of iritis/ hyphaema. 9 patients had corneal tear. Out of this, 6 patients had corneal scleral tear and 3 patients had isolated corneal tear. All of them were managed surgically.

ANTERIOR CHAMBER:

Hyphaema:

19 patients had hyphaema. Out of this, 9 patients were due to globe rupture. They were managed surgically and anterior chamber wash given. 7 patients had reaccumulation of blood in anterior chamber which was treated with topical Beta blocker eye drop and carbonic anhydrase inhibitor orally.

Remaining 10 patients had hyphaema not associated with a tear. 7

patients had intra ocular pressure greater than 25mm Hg. Out of this 5 patients responded to medical management and 2 patients with intra ocular pressure greater than 50mm Hg even after medical management needed paracentesis.

Angle recession:

1 patient had an angle recession <180 degree. Patient was initially treated outside and had no hyphaema on presentation. Patient was advised regular follow up.

IRIS:

14 patients had traumatic mydriasis out of this 6 patients had sphincter tear extending upto 2-3mm from pupillary margin. 3 patients had traumatic miosis.

Traumatic iritis was seen in 14 patients of which 4 patients had intra ocular pressure > 25mm Hg. They responded to topical steroids and antiglaucoma drugs.

LENS:

20 patients had injury to the lens. This included traumatic cataract, subluxated and dislocated lens.

12 patients had traumatic cataract. Of which 3 patients had associated corneal tear. Of the 3 patients, 2 patients underwent corneal

tear suturing alone initially, cataract extraction with PCIOL implantation was done after the eye became quiet. Post operative vision was 6/12. 1 patient underwent corneal tear suturing and lens extraction with PCIOL implantation in the same sitting. Vision improved to 6/36. Remaining 9 patients with old traumatic cataract underwent cataract extraction and PCIOL implantation. Vision improved to 6/24 to 6/6.

4 patients with subluxated lens underwent cataract extraction alone. Aphakic correction with spectacles was given for 2 patients who had mature cataract in the other eye.

4 patients presented with dislocation of lens. Out of this 3 patients had posterior dislocation of lens which was left as such as the eye was quiet and advised scleral fixation of IOL. One patient with dislocated lens into the anterior chamber and elevated ocular tension was managed with lens removal and anterior vitrectomy.

GLOBE RUPTURE:

9 patients presented with corneo scleral tear close to the limbus. 4 were adult males, 4 were adult female and 1 patient was a male child. Out of the 9 patients 7 patients presented with superonasal tear extending 5 to 7 mm from limbus. 2 patients had inferotemporal tear. Vision on presentation was PL in all the patients. 1 patient had an occult

superonasal tear with phacocoele. Tear was suspected because of the presence of large hemorrhagic chemosis.

Corneoscleral suturing was done in all the patients. None of the eye was enucleated. Vision on followup after 1 month was hand movements in 7 patients and no perception of light in 2 patients due to phthisis bulbi.

POSTERIOR SEGMENT INJURIES

VITREOUS HAEMORRHAGE:

5 patients presented with vitreous hemorrhage. B scan was done for all the patients and none of the patients had retinal detachment. Patients were treated with oral prednisolone to decrease the chance of fibrosis of the vitreous. All the patients were advised observation for 6 months.

RETINA:

Retinal tear:

1 patient had a horse shoe shaped retinal tear involving supero nasal quadrant. Prophylactic scleral buckling and cryopexy was done.

Retinal detachment:

3 patients presented with retinal detachment with old tear. Out of this 2 patients had inferior retinal detachment and 1 had superonasal detachment. All the patients had a history of blunt injury to the eye 4 - 6 months back and they were treated outside. They presented with

decreased vision. Scleral buckling with cryopexy was done in all the 3 patients. Anatomical repositioning of retina was good in all the 3 patients. Visual outcome was 6/36 in 1 patient and 2 patients with macular involvement had vision hand movements on followup after 1 month.

Retinal dialysis:

1 patient had inferior retinal dialysis which was diagnosed 1 month after blunt trauma. Patient presented with vision 3/60 with macula on. Anterior segment was normal. Indirect ophthalmoscopy showed a large inferior dialysis. Scleral buckling and cryopexy was done. On follow up after 3 months visual acuity was 6/36.

Optic nerve head avulsion:

1 young adult male had severe injury to the face and head following RTA. He presented with no perception of light. Examination revealed a afferent pupillary defect of right eye. Fundus showed a large retinal hemorrhage involving the optic disc. Ultrasound B scan showed optic nerve head avulsion of right eye.

Traumatic optic neuropathy:

13 patients presented with traumatic optic neuropathy. Out of the 13 patients 11 patients were male and 2 were female. All the patients developed loss of vision immediately after injury. On examination there was abrasion over lateral part of the eyebrow in all the patients. Vision

was 2/60 to no perception of light. Afferent pupillary defect was present. 11 patients had normal fundus, 1 had macular oedema and the other had purtscher's retinopathy.

Out of the 13 patients of suspected optic nerve injury CT scan orbit was done for 9 patients none of the patients with suspected optic nerve injury showed fracture of the optic canal. All the patients had indirect injury to the optic nerve and were treated with intravenous methyl prednisolone 500mg bd for 3 days. None showed improvement in vision.

Mode of injury and presentation of patients with optic nerve injury is represented in the following table

MACULA:

Macular oedema:

6 patients presented with macular oedema. They were of the age group 12 to 30 years. Out of the 6 patients, 4 patients were adult males. 1 was an adult female and 1 was a female child. 5 patients had vision on presentation of 3/60 to 6/60. Follow up vision after 3 months showed improvement to 6/24 to 6/6. 1 patient who had an associated indirect optic nerve injury presented with vision-HM and on follow up after 1 month vision deteriorated to NO PL.

Macular hole:

1 patient presented with macular hole with a history of injury to right eye with a log 4 years back. Vision on presentation was 3/60. Fundus showed full thickness macular hole with proliferative retinopathy.

CHOROID:

4 patients presented with choroidal tear. Out of the 4 patients 2 patients presented immediately after injury. There was an associated subretinal hemorrhage, on resorption of hemorrhage choroidal tear was seen temporal to macula. There was no visual impairment. They were

advised regular follow up for the possibility of choroidal neo vascularisation.

1 patient presented with choroidal tear 3 months after injury. Tear involved the fovea. Visual acuity was 3/60. Yet another patient presented with proliferative vitreoretinopathy with macular hole with a history of blunt trauma 4 years back. Visual acuity was hand movement.

Purtscher's Retinopathy:

2 patients presented with purtscher's retinopathy following RTA. And an adult female had bilateral purtscher's retinopathy. Vision on presentation was 1/60 in both the eyes. Patient was treated with oral prednisolone. There was no visual improvement during follow up to three months. Other patient had purtscher's retinopathy of one eye associated with indirect optic neuropathy vision on presentation was NO PL.

Nerve palsy:

6 patients had ocular motor nerve palsy with normal vision following RTA. Out of which 4 patients had III nerve palsy and 2 patients had VI nerve palsy. Mode of injury in all the patients were RTA. They were managed with steroids. Follow up showed complete recovery in all the patients with VI nerve palsy. 1 patient with III nerve palsy had no improvement and 3 patients with III nerve palsy had partial recovery at

the end of 3 months.

Blow out Fracture:

6 patients presented with blow out fracture of the floor of orbit. Out of the 6, 5 patients had mild elevation restriction with infra orbital anesthesia and all of them were confirmed with CT scan orbit. 1 female child had enophthalmos $> 2\text{mm}$ on presentation. CT scan left orbit showed prolapse of orbital contents into maxillary sinus. She was referred to plastic surgery department for correction post operative recovery was good. Other patients were treated with intravenous steroids. Elevation of globe returned normal and none of them developed enophthalmos.

Lateral wall fracture:

7 patients presented with lateral wall fracture. Out of this 5 had indirect optic nerve injury. 1 had optic nerve avulsion. Other 2 had no ocular damage and they were referred to plastic surgery department.

Medial wall fracture:

1 patient presented with medial wall fracture. Patient had periorbital ecchymosis, subcutaneous emphysema with subconjunctival hemorrhage. CT scan showed medial wall fracture with intra orbital air. Patient was advised not to blow the nose and antibiotic was given.

Eye lid:

Periorbital ecchymosis was seen in 43 patients. 1 patient had eyelid

hematoma involving right upperlid. Surgical evacuation of hematoma was done.

B Scan:

Ultra sound B Scan orbit was done for 13 patients. 5 patients with traumatic cataract with no view of posterior segment underwent B scan orbit and posterior segment was normal in all the patients. One patient with a large preretinal hemorrhage over optic disc proved to be an optic nerve avulsion. B scan was done in all the 5 patients with vitreous hemorrhage. None of them showed retinal detachment. One patient with posterior dislocation of lens had no posterior segment trauma. One patient had retinal detachment.

CT scan orbit

CT scan orbit was done in 22 patients who sustained injury by RTA. Table shows the CT scan orbit finding.

S. No	CT scan orbit	Diagnosis
1	Fracture frontal bone	Indirect optic neuropathy
2	Fracture lateral wall	Indirect optic neuropathy
3	Fracture roof	SCH and Periorbital ecchymosis
4	Fracture floor and zygoma	Periorbital ecchymosis
5	Fracture floor	Blow out fracture
6	Fracture lateral wall	Intra orbital air
7	Fracture lateral wall	Optic nerve avulsion
8	Fracture roof	Indirect optic neuropathy
9	Fracture medial wall	Intra orbital air
10	Normal	Periorbital ecchymosis

11	Normal	Periorbital ecchymosis
12	Fracture Floor	Blow out fracture
13	Normal	SCH and Periorbital ecchymosis
14	Normal	Indirect optic neuropathy
15	Fracture floor	Indirect optic neuropathy
16	Fracture lateral wall	Indirect optic neuropathy
17	Fracture floor	Blow out fracture
18	Normal	Periorbital ecchymosis
19	Fracture floor	Blow out fracture
20	Fracture floor	Blow out fracture
21	Upper lid hematoma	Hematoma lid
22	Fracture lateral wall	Indirect optic neuropathy

DISCUSSION

The study on 110 patients with blunt injury to the eye included patients with trivial to gross external injuries, with mild to gross visual loss.

In this study male patients were 83 and female patients were 27. Male: Female in our study is 3:1. According to a study conducted by Dr. KUHN et al¹⁵ has incidence of male to female of 4:1.

Incidence of blunt trauma was more in male probably due to increased exposure of men to RTA and work place accidents. Age group in which blunt trauma to eye was common was 21 to 30 years. They constituted 30.9% of total injuries. High incidence of trauma to eye was at the third decade. This is probably due to increased sports and work related exposure in this age group. Incidence was less in the extremes of life because of less exposure to accident prone environment.

Road traffic accidents constituted 36.36% of the total injuries. More number of RTA patients in our study is probably due to bad road condition and poor traffic control in our country. Work related injuries were 33.36%. This is probably due to lack of eye protective measures at work place.

Injury to left eye was more common in our study.

Out of the 110 patients, 8 patients had no perception of light during presentation. They were due to optic nerve injury. 5 patients with optic nerve injury had vision PL to 6/60 on presentation, who on followup after 1 month had no perception of light. 9 patients with globe injury had perception of light on presentation. Vision improved to hand movements to 2/60 after suturing. This poor visual outcome was due to lens extrusion, vitreous loss and retinal damage. Patients with vitreous hemorrhage had PL on presentation and vision on follow up after 3 months was 3/60. This was probably due to damage to retina.

12 patients presented with intra ocular pressure greater than 25mm Hg. Out of the 7 patients with increased ocular tension due to hyphema responded to topical beta blockers and cycloplegics and 2 patients failed to respond to medical treatment and paracentesis was done. 4 patients with increased ocular tension due to iritis responded to medical management. 1 patient with increased ocular tension due to anterior dislocation of lens underwent lens removal. 9 patients with globe rupture regained normal intra ocular tension after suturing. So water tight wound closure is important.

51 patients had sub conjunctival hemorrhage. All the patients underwent detailed examination to rule out the possibility of occult scleral rupture, fracture orbit and posterior segment injuries. Out of the 51

patients, 16 patients had orbital wall fracture, 40 patients had posterior segment injuries, and 1 had occult scleral rupture. So it is very important to do a detailed examination in any patient with traumatic subconjunctival hemorrhage. 5 patients had conjunctival chemosis associated with subconjunctival hemorrhage. Out of this one had a large occult scleral tear beneath the hemorrhagic chemosis which was surgically corrected. So any patient of hemorrhagic chemosis needs exploration to rule out occult scleral tear, carotid cavernous fistula.

Out of the 6 patients with conjunctival tear, 1 patient had tear of more than 1cm which was sutured, taking care not to include tenon's capsule within the suture.

8 patients had corneal abrasion. They were treated with topical antibiotics and cycloplegics. Eye patching was done in all the patients. According to a study at Massachusetts 1995^{J16} patching is not required to treat corneal abrasion. But we did patching for all the patients because in our country, due to poor hygienic condition chances of infection is more. So it is better to do patching for 24 hours. Topical NSAID drops were added for few patients who had severe pain. Topical NSAID are effective analgesic for corneal abrasion according to a study at Ottawa-2005^{J17}. Topical antibiotic eye ointment was not advised because of delayed wound healing. This was proved in a study at UK-1999^{J18}. This study

concludes that high prevalence of recurrent symptoms in night ointment application.

9 patients presented with traumatic hyphema associated with corneo scleral tear for which suturing of the tear and anterior chamber wash was given. Out of this 7 patients had reaccumulation of blood and was treated conservatively. 10 patients had hyphema due to closed globe injury. Out of this 7 patients had elevated intra ocular pressure which was treated with topical beta blocker eye drops and carbonic anhydrase inhibitors and advised bed rest. 5 patients responded to this management and 2 patients needed paracentesis. Timely paracentesis is important to prevent permanent optic nerve damage and corneal blood staining.

3 patients presented with traumatic cataract with corneal tear. 2 patients underwent corneal tear suturing initially, and secondary cataract extraction with PCIOL implantation after the inflammation subsided. Vision was 6/12. 1 patient underwent corneal tear suturing and primary cataract extraction and PCIOL implantation at the same sitting. Vision was 6/36. 7 adults with traumatic cataract under went cataract extraction with IOL implantation. Followup vision was 6/24 to 6/6. patients with secondary IOL implantation had better visual outcome than primary IOL implantation. Secondary cataract removal is always better because of better visibility and better IOL calculation.

2 patients with traumatic cataract were children around 10 years. Needling aspiration with PCIOL implantation was done for both the patients. Post operative vision was 6/12. But on followup after 3 months vision was 6/60 due to PCO. According to a study at India 1998^{J20} incidence of PCO was 92%. They advised to clear the axial posterior capsule at the time of surgery or soon after wards.

During cataract surgery ruptured anterior capsule was identified with the help of trypan blue, studied by Ohta 1998^{J19}.

4 patients presented with subluxuated lens. Out of this 2 patients were in the age group of 50 to 60 years and other 2 patients were in the age group of 20 to 30 years. 4 patients with dislocation of lens were in the age group of 50 to 60 years. Incidence of zonular dehiscence was more above the age of 50 years. This was similar to a study by Joel ^{J21}. In the presence of zonular dehiscence in young adults with trivial injury, systemic diseases like marfans, homo cystinuria to be ruled out.

9 patients presented with globe rupture. Of this 8 were adults and 1 was a male child of 5 years. 6 patients were male and 3 were female. Globe injury was more common in male than in female and superonasal tear was more common. This is similar to a study by Cherry et al^{J22}. Superonasal tear is more common due to injury to unprotected infero temporal quadrant.

Absence of hyphema, rupture less than 9mm and use of cryo therapy or diathermy at the time of closure are all good prognostic signs Cherry et al^{J22}.

Study at SLOVENIA^{J23} states that half of the patients with good visual outcome after globe rupture were the one's treated with pars plana vitrectomy. Relative afferent pupillary defect as a functional test is a good predictor for visual outcome.

5 patients with vitreous hemorrhage had B scan orbit done. None of the patients showed retinal detachment. All the patients were kept under observation for spontaneous resorption of hemorrhage. Visual acuity on follow up after 3 months was only 3/60.

1 patient with superonasal retinal detachment had visual acuity 6/36 after scleral buckling and cryopexy. 3 patients with retinal detachment and tear and underwent surgery. Vision was HM because of delayed surgery after the injury. 1 patient had isolated retinal tear who underwent prophylactic scleral buckling and cryopexy. 1 patient with retinal dialysis underwent scleral buckling and cryopexy with vision 6/36 after 3 months.

Study at Northern Ireland 1991^{J24} states that visual prognosis is good when retinal breaks and detachment were diagnosed within 6 weeks

of injury. Retinal detachment was a feature of necrotic retinal breaks while inferior dialysis led to slow accumulation of sub retinal fluid and had better visual prognosis.

4 patients had choroidal tear. Of this 2 patients had acute choroidal tear associated with sub retinal hemorrhage. Vision was 6/60. On resorption of hemorrhage tear was temporal to macula. Patients were advised regular followup for the early diagnosis of choroidal neovascularisation which is the common complication of choroidal tear. Remaining 2 patients were old choroidal tear of which 1 had foveal involvement. In our study 3 patients had tear temporal to fovea and 1 patient had foveal involvement. Male : Female is 3:1, age group 30 to 40 years. This is similar to a study at USA 2006.^{J25}

5 patients presented with macular oedema. Vision on presentation was 6/60 and patients were treated conservatively. Followup vision after 1 month was 6/24 to 6/6.

13 patients presented with traumatic optic neuropathy. Mode of injury in 11 patients were road traffic accidents and in 2 patients due to fall by self. Visual loss occurred immediately after injury and it was gross. On examination all the patients had laceration over eyebrow and lateral side of forehead, which is the most common site of injury leading to indirect optic nerve damage. Relative afferent pupillary defect was

noticed in all the patients. CT scan orbit was done in 9 patients with optic nerve injury of which 6 patients had fracture orbital walls, 3 patients had normal CT orbit. None of the patient had fracture of the optic canal or hematoma of optic nerve sheath. All the patients had indirect injury to the optic nerve. There was no co relation between CT scan orbit and optic nerve injury. This is similar to a study at USA 1984^{J26} which states that there is no co-relation between abnormalities detected by CT scan and optic nerve injury. Optic canal fracture is not necessary for injury to optic nerve. But still patients with sudden visual loss after head trauma should undergo CT scan of optic canal region to look for any direct injury to optic nerve.

All the patients were treated with intravenous methyl prednisolone but none had visual improvement.

Study at USA 1996^{J27} on traumatic optic neuropathy concludes that there was no significant difference in improvement was found among patients treated with corticosteroids alone, with surgical decompression alone or both. Recovery was related to the severity of initial injury.

2 patients presented with purtscher's retinopathy. Out of this, 1 patient had bilateral purtscher's retinopathy. Vision in both eyes were 1/60. Other patient had unilateral purtscher's retinopathy along with

indirect optic neuropathy. Mode of injury in both the patients were road traffic accidents. Both the patients developed deterioration of vision. There is no proven therapy for purtscher's retinopathy.

4 patients had 3rd nerve palsy with normal vision. On followup, 3 patients with 3rd nerve palsy showed partial recovery and 1 patient had no recovery. None of the patients developed aberrant regeneration over 3 months followup.

2 patients presented with 6th Nerve Palsy. Complete recovery occurred in both the patients at the end of 4 months. Road traffic accident was the cause for nerve palsy in all the patients.

6 patients had blow out fracture of orbital floor. 5 patients presented with infra orbital anaesthesia, restriction of elevation of eye ball. CT scan orbit showed floor fracture without prolapse of orbital contents. Oral steroids were given. On followup none of the patient developed enophthalmos.

1 patient with blow out fracture enophthalmos on presentation and CT scan orbit coronal view showed prolapse of orbital contents into the maxillary sinus. Patient was referred to plastic surgery department for floor repair. Post operative recovery was good.

B scan was done pre operatively in patients with traumatic cataract to rule out posterior segment abnormality and to assess the posterior

capsule integrity for IOL implantation. Vitreous hemorrhage is another indication for B scan to look for any retinal damage. Ultrasound on followup is helpful in patients with vitreous hemorrhage for early diagnosis of retinal detachment. Functional success of retinal detachment surgery is high only in patients with early surgery.

CT scan was done in 22 patients with RTA. It was done in patients with marked loss of vision immediately after vision to rule out the possibility of direct optic nerve injury. The only condition with blunt injury eye in which emergency CT scan orbit is required is suspected optic nerve injury. CT scan orbit was done in suspected orbital floor fracture with severe restriction of elevation of globe and enophthalmos to study the extent of bony defect and assess the amount of prolapse of orbital contents into the maxillary sinus. This helps to decide about surgery.

SUMMARY

The study on 110 patients with blunt injury to the eye had 83 male patients and 27 female patients.

Male: Female was 3:1

Age group with more incidence of blunt trauma was 21 - 30 years.

Road traffic accidents had highest incidence among all injuries.

Among non-RTA patients work related injuries were more common.

Eye most commonly involved in our study is left eye.

Vision on presentation was from no perception of light to 6/6. 8 patients had no perception of light due to optic nerve injury. On follow up 17 patients had no perception of light due to optic nerve injury of which nine patients on presentation had vision PL to 2/60.

20 patients had vision, perception of light to hand movements on followup. They were due to vitreous hemorrhage, retinal detachment, purtscher's retinopathy, choroidal tear, macular hole and globe rupture. Rest of the patients showed visual improvement.

12 patients had intraocular pressure greater than 25 mm Hg. All the patients responded to medical or surgical management. None of them developed chronic elevation of intraocular pressure.

Sub conjunctival hemorrhage and chemosis subsided completely.

conjunctival tear healed without any complication.

8 patients had corneal abrasion. 7 patients responded to topical anti-biotic drops. 1 patient with recurrent erosion responded to bandage contact lens. 12 patients of corneal oedema subsided with management of associated iritis/hyphema. None of them developed permanent corneal oedema.

Out of 19 patients with hyphaema, 9 patients responded after surgical intervention. 8 patients responded to medical management and 2 patients needed paracentesis. None of the patient developed corneal blood staining or optic nerve head damage.

Iritis was seen in 14 patients. 10 patients responded to topical steroids. 4 Patients developed chronic iritis. This is probably due to poor compliance of patients.

Traumatic miosis subsided after the acute stage of injury.

Traumatic mydriasis was managed conservatively. None of them needed sphincter tear suturing.

9 patients with isolated traumatic cataract had visual acuity 6/24 to 6/12 after cataract extraction and PCIOL implantation.

3 patients of traumatic cataract with corneal tear had visual acuity of 6/36 after corneal tear suturing and PCIOL implantation.

4 patients with subluxuated lens had lens removal and were advise secondary IOL implantation because of diplopia with glasses. 4 patients

of dislocated lens were advised secondary IOL implantation.

9 patients of globe rupture had corneo scleral tear suturing. None of them were enucleated. 7 patients had vision hand movements. 2 patients developed phthisis bulbi.

5 patients presented with vitreous hemorrhage. All the patients were kept under observation. Vision on follow up after 3 months was 3/60.

1 patient with isolated retinal tear under went prophylactic scleral buckling and cryopexy. Vision on follow up after 1 month was 6/12.

3 patients with retinal detachment under went scleral buckling and cryopexy. Anatomical reapposition of retina was good. Functional recovery was poor. One patient with inferior retinal dialysis under went scleral buckling and cryopexy. Visual improvement was good.

One patient had optic nerve avulsion following severe road traffic accident and had no perception of light.

13 patients had indirect optic neuropathy they were treated with intravenous methyl prednisolone. None of them showed visual improvement. Vision deteriorated to no perception of light.

6 patients with macular oedema had vision 6/60 to 3/60 on presentation. Vision on follow up was 6/24 to 6/6.

2 patients had acute choroidal tear temporal to macula. They had no visual impairment on follow up after 3 months. One patient had old

tear involving fovea with vision 3/60. Choroidal neovascularisation was not detected during the study period.

1 patient with bilateral purtscher's retinopathy had severe visual impairment on presentation. Vision deteriorated further.

2 patients with 6th nerve palsy showed complete recovery. One patient with 3rd nerve palsy had no recovery and 3 patients had partial recovery and none showed aberrant regeneration at the end of 3 months.

Among the 6 patients with blow out fracture only one patient needed surgical repair.

5 patients with lateral wall fracture had indirect optic neuropathy. 1 had optic nerve avulsion. And 1 patient had no ocular damage.

One patient with medial wall fracture was treated with high dose of antibiotic and referred to plastic surgery department.

One patient with a large upperlid hematoma was surgically evacuated.

CT scan was useful to rule out direct optic nerve injury and assess the blow out fracture.

B scan helped in preoperative assessment and in follow up.

CONCLUSION

In our Study,

- Blunt trauma was more common in males.
- Most common age group was 3rd decade.
- Road traffic accidents constituted large number of blunt injuries.
- Road traffic accidents had a high rate of visual loss due to optic nerve injury.
- Work related injuries were common among non RTA patients.
- Few patients developed chronic iridocyclitis due to poor compliance.
- Visual loss was not severe in anterior segment lesion.
- Trivial anterior segment injury had vision threatening lesion in the posterior segment. So it is very important to examine the posterior segment in all the patients with injury to the eye.
- Secondary cataract extraction had comparatively better visual outcome than primary cataract extraction.
- Posterior segment lesion led to total visual loss due to retinal detachment, vitreous hemorrhage, choroidal tear and optic nerve injury.
- Globe rupture led to severe visual loss.
- CT scan is important to rule out direct optic injury and timely intervention and in surgical repair in blowout fracture.

- B scan helped in patients with traumatic cataract to rule out posterior segment pathology and assess posterior capsule integrity to plan IOL implantation.

Since blunt injury eye leads to severe visual impairment its prevention is better. Creating awareness for eye protection is important. Better road traffic rules and educating the public regards protection of eye at work places. Creating awareness among school children regarding eye protection during sports activity is important.

BIBLIOGRAPHY

Text Books

1. Albert & Jakobiec principles and practice of ophthalmology 2nd Edition pages 5179 to 5227.
2. Principles and practice of ophthalmology Peyman – Sanders – Goldberg pages 2425 to 2509.
3. Parson's diseases of the eye. Nineteenth Edition pages 402 to 411.
4. American Academy of ophthalmology orbit, Eyelids and Lacrimal system 2006 – 2007, pages 97 – 108.
5. Duke Elder S. System of ophthalmology Vol. XIV part I, mechanical injuries.
6. Duane's clinical ophthalmology vol 3, chapter 31.
7. American Academy of ophthalmology Retina and vitreous 2006 – 2007 pages 289 – 302.
8. Stallard's Eye surgery seventh Edition. M.J. Roper-Hall pages 384 to 415.
9. American Academy of ophthalmology. External disease and cornea. Section 8: pages 403 to 417.
10. American Academy of ophthalmology. Section 11: lens, cataract pages 54, 55, and 225 to 228.

Journals

1. A system for classifying mechanical injuries to the eye. American journal of ophthalmology vol 123 pages 820 – 831.
2. Courville CB: coup contre-coup mechanism of craniocerebral injuries Arch surg 45: 19, 1942.
3. Wolter JR coup contre-coup mechanism of ocular injuries. AmJ

- ophthalmology 56: 785, 1963.
4. Zigelbaum B, Tostanoski JR, Hochman M.A Hersh PS. Combined Lignocaine and proparacaine anaesthetic abuse. AmJ emerg Med 12: 96, 1994.
 5. Kenyon KR: Recurrent corneal erosion. Pathogenesis and therapy. Int ophthalmol clin 19: 169, 1979.
 6. MC. Lean, EN. Mac Rae, SM. Rich Lf. Recurrent erosion. Treatment by anterior stromal puncture. Ophthalmology 93:784, 1986.
 7. Jeboul B.K. Hacob JL. Barsoum Homsay.M et al: Clinical evaluation of aminocaproic acid for managing traumatic hyphema in India ophthalmology 102: 1646, 1995.
 8. MC Getrick JJ, Jampol L.M Goldberg MF et al aminocaproic acid decreases secondary Hemorrhage after traumatic hyphema. Arch ophthalmol 101: 1031, 1983.
 9. Paton D. Craig J: Manag. of iridodialysis. Ophthalmic surgery 4: 38, 1973.
 10. Mermound A Salmon JF Barron A, et al. Surgical management of post traumatic angle recession glaucoma ophthalmology 100: 634, 1993.
 11. Epstein DC. Jedziniak JA Grant: Obstruction of aqueous outflow by lens particles & heavy mol-wt soluble lens proteins invest ophthalmol 17: 272, 1978.
 12. Andreou P.Clarke MP Feency P. A case of traumatic retinal avulsion at the optic nerve head. Br J ophthalmol 80: 575, 1996.
 - 13.Scott J D. Retinal dialysis. Trans ophthalmol SOC UK 97: 33, 1977.
 - 14.Pulido JS Blair NP. The blood retinal barrier in Berlin's oedema. Retina 7: 233, 1987.

15. May Dr. KUHN FP, Morris Re et al: The epidemiology of serious eye injuries from the United States injury registry. Graefes Arch clin Exp ophthalmol 238: 53, 2000.
16. Corneal abrasion patching study group. Massachusetts Eye & Ear Infirmary. HARVARD Medical school Boston U.S.A ophthalmol 1995 Dec.
17. Topical NSAID drugs for corneal abrasion. Meta analysis. Department of Emerg medicine Ottawa Hospital CANADA ACAD Emerq Med 2005 May.
18. Topical ointment does not prevent rec. symptoms in corneal abrasion Br. J. ophthalmol 1998 Sep. Department of ophthalmology Leicester Royal infirmary.
19. Aravindh Eye hospital & post graduate institute of ophthalmology Madurai India. Br J ophthalmol 1998 Aug.
20. Staining of anterior capsule for CCC in eyes with white cataract. Horiguchi M. Miyake K. Ohta I. Ito Arch ophthalmol 1998.
21. Blunt trauma to aging Eye. Joel D. Stitzel Ph D. Gail A. Hansen Arch ophthalmol 2005 123: 789 – 794.
22. Indirect traumatic rupture of globe PM cherry Arch ophthalmol vol 96, No 2, Feb 1978.
23. To establish surgery results and establish prognostic factors of open globe injury. Eye clinic Ljubljana University. Medical centre Slovenia CROAT Med J 2004 June.
24. Traumatic retinal detachment. Department of ophthalmology Royal Victoria Hospital Belfast, Northern Ireland. BJO 1991 June.
25. Ament CS Zack DN Lane. Predictors of visual outcome and choroidal neovascular membrane formation after Traumatic choroidal rupture. Arch ophthalmol Jul 2006.
26. Computed Tomographic evaluation of optic canal in sudden

traumatic blindness. AmJ ophthalmol 1984 Dec.

27. Traumatic optic Neuropathy. Meta analysis Washington Medical Centre. Seattle U.S.A Arch Ophthalmol Head Neck Surg 1996 April.

PROFORMA

A STUDY ON BLUNT INJURY TO THE EYE AND ITS

MANAGEMENT

1. Serial Number :
2. Hospital Number :
3. Name :
4. Age :
5. Sex : M ☐ F ☐
6. Address :
- Rural ☐ Urban ☐
7. Occupation : Agriculture ☐ Industrial ☐
Others ☐
8. Mode of injury :
- i) Accident : Assault ☐ RTA ☐
- ii) While at work :
- iii) Not at work :
9. Affected Eye : Right ☐ Left ☐
10. Agents Causing Injury : Stick ☐ Stone ☐ Ball ☐
Fire ☐ Metal ☐ Other ☐
11. Date and time of injury :

12. Date First Seen :
13. Nature of First Aid given :
14. Ocular status before accident:
15. Vision : Right Eye Left Eye
16. Ocular Movements :
17. Conjunctiva and Sclera :
18. Cornea :
19. Anterior Chamber :
20. Iris and Ciliary Body :
21. Pupil :
22. Lens :
23. Posterior Segment :
 - a) Vitreous :
 - b) Retina :
 - c) Choroid :
24. Optic Nerve :
25. Refraction :
26. Tension :
27. Gonioscopy :
28. Investigation :
 - X –Ray Orbit
 - Skull :
 - Ultrasound B scan Orbit :
 - CT Scan Orbit :

Others

:

Diagnosis

Sequae

Followup